Application No.: 10/076,204 Filing Date: February 13, 2002

Docket No.: 955-16

Page 2 of 6

## IN THE CLAIMS

Please cancel Claims 31-32 without prejudice.

Please amend Claim 1 as follows:

- 1. (Currently Amended) A method for reducing cardiac dysfunctions in a human in need thereof wherein the cardiac dysfunction is due to a pathological excess of norepinephrine release, the method comprising a administering to the human an effective amount of a selective histamine H<sub>3</sub> receptor agonist.
- 2. (Original) The method according to claim 1, wherein the cardiac dysfunction is associated with myocardial ischemia or myocardial infarction.
- 3. (Original) The method according to claim 1, wherein the cardiac dysfunction is arrhythmia, fibrillation, platelet activation and aggregation, thrombus formation, coronary spasm, sudden cardiac death or cardiac failure.
- 4. (Original) The method according to claim 1, wherein the selective histamine  $H_3$  receptor agonist is R-( $\alpha$ )-methylhistamine, imetit, immepip, immepyr, 4-(1H-4-imidazolylmethylene)1-methylpiperidine, S- $\alpha$ -chloromethylhistamine, cyclopropylhistamine, SKF 91606, Sch 50971, VUF 4864.
- 5. (Original) The method according to claim 1, wherein the selective histamine H<sub>3</sub> receptor agonist is administered after the onset of myocardial ischemia and/or myocardial infarction.
- 6. (Original) The method according to claim 1, wherein the selective histamine H<sub>3</sub> receptor agonist does not act on the central nervous system.
- 7. (Original) The method according to claim 1, wherein the selective histamine H<sub>3</sub> receptor agonist does not cross the blood brain barrier.



Application No.: 10/076,204 Filing Date: February 13, 2002

Docket No.: 955-16

Page 3 of 6

- 8. (Original) The method according to claim 1, wherein the histamine H<sub>3</sub> receptor is on a cardiac sympathetic nerve ending.
- 9. (Original) The method according to claim 1, wherein the histamine H<sub>3</sub> receptor agonist reduces norepinephrine release from a cardiac sympathetic nerve ending.
- 10. (Original) The method according to claim 1, wherein the reduction in norepinephrine release is specifically antagonized by an H<sub>3</sub>R antagonist.
- 11. (Original) The method according to claim 1, wherein the H<sub>3</sub>R antagonist is Thioperamide or Clobenpropit.
- 12. (Original) The method according to claim 1, wherein the histamine  $H_3$  receptor agonist inhibits the  $Na^+/H^+$  exchanger.
- 13. (Original) The method according to claim 12, wherein the histamine H<sub>3</sub> receptor agonist inhibits the Na<sup>+</sup>/H<sup>+</sup> exchanger on a cardiac sympathetic nerve ending.
- 14. (Original) The method according to claim 1, wherein the histamine H<sub>3</sub> receptor agonist modulates the concentration of intracellular sodium.
- 15. (Original) The method according to claim 1, wherein the histamine H<sub>3</sub> receptor agonist modulates the concentration of intracellular calcium.
- 16. (Original) The method according to claim 15, wherein the histamine H<sub>3</sub> receptor agonist modulates the concentration of intracellular calcium by inhibiting the activity of an N-type Ca<sup>2+</sup> channel.



Application No.: 10/076,204 Filing Date: February 13, 2002

Docket No.: 955-16

Page 4 of 6

- 17. (Original) The method according to claim 1, wherein the histamine H<sub>3</sub> receptor agonist is delivered in combination with at least one other agent in the treatment of cardiac dysfunction.
- 18. (Original) The method according to claim 17, wherein the other agent is one or more of the following: a  $\beta$ -blocker, a Ca<sup>++</sup>-channel blocker, an anti-arrhythmic, an ACE inhibitor and an angiotensin receptor antagonist.
- 19. (Original) A method for inhibiting the Na<sup>+</sup>/H<sup>+</sup> exchanger in a human having a cardiac dysfunction, the method comprising administering to the human an effective amount of a selective histamine H<sub>3</sub> receptor agonist.
- 20. (Original) The method according to claim 19, wherein the cardiac dysfunction is myocardial ischemia or myocardial infarction.
- 21. (Original) The method according to claim 19, wherein the cardiac dysfunction is arrhythmia, fibrillation, platelet activation and aggregation, thrombus formation, coronary spasm, sudden cardiac death or cardiac failure.
- 22. (Original) The method according to claim 19, wherein the selective histamine  $H_3$  receptor agonist is R-( $\alpha$ )-methylhistamine, imetit, immepip, SKF 91606 or Sch 50971.
- 23. (Original) The method according to claim 19, wherein the selective histamine H<sub>3</sub> receptor agonist is administered after the onset of myocardial ischemia and/or myocardial infarction.
- 24. (Original) The method according to claim 19, wherein the selective histamine H<sub>3</sub> receptor agonist does not act on the central nervous system.
- 25. (Original) The method according to claim 19, wherein the selective histamine H<sub>3</sub> receptor agonist does not cross the blood brain barrier.

Application No.: 10/076,204 Filing Date: February 13, 2002

Docket No.: 955-16

Page 5 of 6

- 26. (Original) The method according to claim 19, wherein the histamine H<sub>3</sub> receptor is on a cardiac sympathetic nerve ending.
- 27. (Original) The method according to claim 19, wherein the histamine H<sub>3</sub> receptor agonist inhibits norepinephrine release from cardiac sympathetic nerve endings.
- 28. (Original) The method according to claim 19, wherein the histamine H<sub>3</sub> receptor agonist modulates the concentration of intracellular sodium.
- 29. (Original) The method according to claim 19, wherein the histamine H<sub>3</sub> receptor agonist is delivered in combination with at least one other agent in the treatment of cardiac dysfunction.
- 30. (Original) The method according to claim 19, wherein the other agent is one or more of the following: a  $\beta$ -blocker, a Ca<sup>2+</sup>-channel blocker, an anti-arrhythmic, an ACE inhibitor and an angiotensin receptor antagonist.

Claims 31-32 (Cancelled).

